SIAM J. APPL. MATH.
Vol. 57, No. 4, pp. 1082–1094, August 1997

010

BEHAVIOR CHANGES IN SIS STD MODELS WITH SELECTIVE MIXING*

JAMES M. HYMAN† AND JIA LI‡

Abstract. We propose and analyze a heterogeneous, multigroup, susceptible-infective-susceptible (SIS) sexually transmitted disease (STD) model where the desirability and acceptability in partnership formations are functions of the infected individuals. We derive explicit formulas for the epidemic thresholds, prove the existence and uniqueness of the equilibrium states for the two-group model and provide a complete analysis of their local and global stability. We then investigate the effects of behavior changes on the transmission dynamics and analyze the sensitivity of the epidemic to the magnitude of the behavior changes. We verify that if people modify their behavior to reduce the probability of infection with individuals in highly infected groups, through either reduced contacts, reduced partner formations, or using safe sex, the infection level may be decreased. However, if people continue to have intragroup and intergroup partnerships, then changing the desirability and acceptability formation cannot eradicate the epidemic once it exceeds the epidemic threshold.

Key words. STD models, selective mixing, balance constraint, behavior changes, reproductive numbers, endemic equilibrium, local stability, global stability

AMS subject classifications. 34D20, 58F40, 92D30

PII. S0036139995294123

1. Introduction.

STDs, such as AIDS, have spread into nearly all countries of the world. For example, it has been estimated that in some regions of central Africa up to 20% of the population is infected by human immunodeficiency virus (HIV) and that in the Bronx in New York City 13% of men and 7% of women aged 25–40 years are HIV positive [15]. To prevent further spread of these epidemics, it is important to understand how these infectious diseases are transmitted.

The transmission dynamics are complex. Many biological and sociological factors are involved. One of major determinants in the spread of STDs is the way that individuals select their sexual partners.

Sexual behavior changes are documented in virtually every survey of homosexual or bisexual men and injection drug users over the past decade [1, 10, 27, 28]. These behavior changes occur as sexually active individuals became more cautious in their sexual activities to avoid infection by an STD such as AIDS. Understanding the effect of these behavior changes can help guide education programs on the prevention of STD transmission.

Some of the analysis of these behavior studies [10, 22, 40] has implied that the reported behavior changes combined with observed reduction in incidence of rectal gonorrhea, HIV infection, and AIDS have been large enough to reduce the rate of transmission of HIV and possibly reduce the rate of HIV transmission below the epidemic threshold. Similar studies have been useful in understanding STD epidemics

*Received by the editors November 6, 1995; accepted for publication March 18, 1996. This research was supported by the Department of Energy under contracts W-7405-ENG-36 and DOE Applied Mathematical Sciences Program contract KC-07-01-01 and was performed while Jia Li was visiting the Theoretical Division and the Center for Nonlinear Studies at the Los Alamos National Laboratory.

http://www.siam.org/journals/siap/57-4/29412.html

†Theoretical Division, MS-B284, Center for Nonlinear Studies, Los Alamos National Laboratory, Los Alamos, NM 87545 (jh@lanl.gov).

‡Department of Mathematical Sciences, University of Alabama in Huntsville, Huntsville, AL 35899 (li@math.uah.edu).

(see, e.g., [2, 26, 29, 30, 34, 37, 38]) and have proved their worth in guiding prevention programs. However, most of the analysis has been for simple compartmental STD models, such as those in [13, 14, 39], and the conclusions are based on the aggregate epidemic threshold conditions. Then, extreme care must be used before applying the conclusions to guiding policy decisions.

To investigate effects of behavior changes on the transmission dynamics, a common approach is to use different sets of parameters and simulate the model for the whole course of the epidemic. Because of the complexity of the transmission dynamics of STDs and difficulty in the mathematical analysis, the models usually assume that the dynamics of partnership formation do not change during the simulation.

In order to study the effects of behavior changes on the STD epidemics, the model must account for the possibly changing dynamics of partnership formation. Although not all of the complicated social and physiological factors involved in human behaviors can be fully accounted for, some crucial aspects are essential to understanding the course of STD epidemics, and they must be modeled. These include behavior changes and the realization that partnership formations are not random but depend upon age and the social and economic backgrounds of the individuals. These attributes can be combined into an aggregate model where partnership formation depends on the mutual desirability and acceptability of the individuals.

In this article we extend and analyze the socially structured, heterogeneous mixing models first proposed by Koopman et al. [24] to model disease transmission. In these models the population is divided into subgroups based on risk levels, age, social behaviors, economic status, ethnic status, and geographic locations. An individual's preference in selecting a partner is determined by desirability and acceptability of individuals in the various groups. We allow the desirability and acceptability of partnership formations to depend upon the fraction of infected individuals in the different groups. That is, people adjust their desirability or acceptability of forming a partnership based on the fraction of the infected population in their potential partner's group.

A goal of this research is to better understand how models with dynamic partnership formation differ from the more traditional models where the number or desirability of partnership formations is constant. By making the partnership formation infection dependent, we can analyze how sensitive the transmission dynamics of the epidemic are to changes in sexual behaviors.

We first formulate the new model and then derive epidemic thresholds for the two-group model and investigate the existence, uniqueness, and local stability of the endemic equilibrium. The global stability of the infection-free equilibrium and the endemic equilibrium is fully analyzed for the two-group model. The effects of several possible behavior changes on the dynamics of the epidemics are finally discussed.

2. Model formulation. The partnership formation is essential in multigroup STD models. Hyman and Stanley [17, 18] formulated risk-based models and pair-formation functions based on the assumption that people in certain groups have priority in choosing their partners. Because the partnership formation must satisfy balance constraints to ensure that the number of partnerships formed by people in group A with people in group B in a given period of time must equal the number of partnerships formed by people in group B with people in group A, complicated mathematical derivations were imposed. Blythe, Busenberg, Castillo-Chavez, and their coworkers [3, 4, 5, 7] formulated similar multigroup models, where the mixing functions or mixing matrices are based on the average activity c_i of individuals in

group i and the average fraction p_{ij} of partners in group j of a person in group i. These formulations lead to complicated integral equations for the mixing functions, which are mathematically difficult to satisfy in an unbiased manner. In both of these approaches, artificial or special acceptance functions were employed to enforce the balance constraints.

This complication can be eliminated using a heterogeneous selective mixing model where the partnership formations are based on the desirability of sexually active individuals and the acceptability and availability of their potential partners. Jacquez, Koopman, Sattenspiel, Simon, and their coworkers developed and analyzed these models in a series of insightful papers [19, 20, 21, 24, 25, 31, 32, 33]. In these selective mixing models, the rates of sexual contacts between individuals fall into categories of discrete population subgroups, and the balance constraints are automatically satisfied. This feature simplifies the analysis and numerical simulation of the models while still accounting for the effects of biased mixing.

We extend the selective mixing model to study the effects of behavior changes on the dynamics of STDs in SIS models, where the population consists of susceptibles and infectives, and it is assumed that the infectives become susceptible again after being diagnosed and treated and that no disease-induced mortality occurs. We further divide the susceptible and infected populations into k groups according to their risk level, S_i and I_i, $i = 1, \ldots, K$, and consider the following simple SIS model:

$$\begin{cases} \dfrac{dS_i}{dt} = \mu(S_i^0 - S_i) - \lambda_i S_i + \gamma_i I_i, \\ \\ \dfrac{dI_i}{dt} = -(\mu + \gamma_i) I_i + \lambda_i S_i, \end{cases} \qquad i = 1, \ldots, K, \tag{2.1}$$

where μ is the natural death rate, γ_i is the rate of recovery for infected individuals in group i, λ_i is the rate of infection, and μS_i^0 is the rate of recruitment into group i.

The formation of partnerships plays an essential role in determining the functional λ_i, which is one of the most important factors in modeling STDs. We define a partnership to be sexual activity between two individuals where the infection can be transmitted (e.g., sexual intercourse). It depends on the desirability of an active individual, the acceptability of his or her potential partners, and the availability of these potential partners. We assume that people in each group behave the same when selecting a partner but that they have biases between groups. In other words, mixing within each group is assumed to be homogeneous, but there is heterogeneous mixing among the groups.

Let q_{ij} be the preference of individuals in group i to have a partner from group j; that is, q_{ij} is the fraction of people in group j with whom an individual in group i desires forming a partnership. Thus q_{ij} describes the desirability of individuals in group i to have a partner from group j. It is also the acceptability of people in group j to people in group i.

Under the condition that enough potential partners are available, the probability p_{ij} that a partnership forms between individuals from group i and group j, or the mutually acceptable rate for partnership formation (see [24]), is

$$p_{ij} := q_{ij} q_{ji}.$$

Define c_i to be the number of social contacts per unit time for a person in group i. The probability that a contact is with a person from group j is $c_j N_j / \sum_k c_k N_k$, where

$N_k = S_k + I_k$. This also characterizes the availability of sexual contacts with partners in group j. Hence, the probability of a partnership forming between individuals from group i and group j is $p_{ij}(c_j N_j / \sum_k c_k N_k)$.

We define β_{ij} to be the probability of disease transmission per contact between an infected partner in group j and a susceptible individual in group i. Under these assumptions, the infection rate of people in group i is

$$\lambda_i = c_i \sum_{j=1}^{K} p_{ij}\beta_{ij} \frac{c_j I_j}{\sum\limits_k c_k N_k},$$

where we assume that $\frac{I_j}{N_j}$ is the probability that a random contact from group j is with an infected individual.

A major advantage of the selective mixing model is that the balance constraints are automatically satisfied because if we let the number of contacts per unit time of people in group i with people in group j be T_{ij}, then it follows from

$$T_{ij} = p_{ij} \frac{c_j N_j}{\sum\limits_k c_k N_k} c_i N_i = p_{ji} \frac{c_i N_i}{\sum\limits_k c_k N_k} c_j N_j = T_{ji}$$

that the balance constraint is always satisfied.

Using the advantages of the selective mixing model, we further assume that the desirability and acceptability depend on the fractions of infected individuals in the populations. This assumption characterizes possible behavior changes of sexually active individuals. More specifically, we assume that the desirability of people in group i having a partner in group j or the acceptability of people in group j to people in group i, q_{ij}, is a decreasing function of the fraction of infected individuals in group j. Then, the mutually acceptable rates for partnership formation can be expressed as

$$p_{ij} = p_{ji} = q_{ij}\left(\frac{I_j}{N_j}\right) q_{ji}\left(\frac{I_i}{N_i}\right),$$

and the infection rates are

$$\lambda_i = c_i \sum_{j=1}^{K} \beta_{ij} q_{ij}\left(\frac{I_j}{N_j}\right) q_{ji}\left(\frac{I_i}{N_i}\right) \frac{c_j I_j}{\sum\limits_k c_k N_k}.$$

3. Epidemic thresholds. The threshold conditions for an STD model [8, 11, 12, 21, 35] specifies when the disease will spread if a small number of infected individuals are introduced into a susceptible population. The threshold conditions can be characterized by the reproductive number R_0, which is determined by the stability of the $2K$ dimensional infection-free equilibrium with components $(N_i, I_i) = (S_i^0, 0)$. In this section we define a reproductive number such that if $R_0 < 1$, the infection-free equilibrium is locally stable, and the epidemic dies out if a small number of infected individuals are introduced. If $R_0 > 1$, the infection-free equilibrium is unstable, and the disease spreads into the population.

System (2.1) is equivalent to

$$\begin{cases} \dfrac{dN_i}{dt} = \mu S_i^0 - \mu N_i, \\ \dfrac{dI_i}{dt} = -(\mu + \gamma_i) I_i + \lambda_i (N_i - I_i). \end{cases} \tag{3.1}$$

Since $\lim_{t\to\infty} N_i = S_i^0$, the limiting system of (3.1) is

$$\frac{dI_i}{dt} = -(\mu+\gamma_i)\, I_i + \lambda_i \left(S_i^0 - I_i\right), \tag{3.2}$$

where

$$\lambda_i = \frac{c_i}{N^0} \sum_{j=1}^{K} q_{ij} \left(\frac{I_j}{S_j^0}\right) q_{ji} \left(\frac{I_i}{S_i^0}\right) \beta_{ij} c_j I_j$$

with $N^0 := \sum_{j=1}^{K} c_j S_j^0$.

The dynamics of (3.1) are qualitatively the same as (3.2) [9, 36], and the stability of the infection-free equilibrium can be determined by the stability of the zero solution of (3.2).

Model (2.1) has been analyzed when there are no infection-dependent behavior changes [16, 21, 35]. The introduction of infection-dependent partnership functions considerably complicates the analysis. In this initial investigation, in order to keep the analysis tractable, we restrict our mathematical analysis to the two-group case. After deriving an explicit formula for the reproductive number, we then investigate the endemic equilibrium and explore the global dynamics of the transmission process.

We denote

$$\begin{aligned} q_{ii}^2 \ \left(I_i/S_i^0\right) &:= w_i\left(I_i\right), \qquad i = 1, 2, \\ q_{12}\left(I_2/S_2^0\right) &:= g\left(I_2\right), \\ q_{21}\left(I_1/S_1^0\right) &:= h\left(I_1\right), \end{aligned} \tag{3.3}$$

where w_i, g, and h are continuously differentiable and decreasing positive functions with

$$w_i, g, h : \mathbb{R} \to \mathbb{R}^+. \tag{3.4}$$

Then the rates of infection are

$$\begin{aligned} \lambda_1 &= \frac{c_1}{N^0}\left(\beta_{11} c_1 w_1\left(I_1\right) I_1 + \beta_{12} c_2 h\left(I_1\right) g\left(I_2\right) I_2\right), \\ \lambda_2 &= \frac{c_2}{N^0}\left(\beta_{21} c_1 h\left(I_1\right) g\left(I_2\right) I_1 + \beta_{22} c_2 w_2\left(I_2\right) I_2\right). \end{aligned} \tag{3.5}$$

The Jacobian matrix of (3.2) at the zero solution has the form of

$$J^0 := \begin{pmatrix} -\mu - \gamma_1 + \dfrac{\beta_{11} p_{11} S_1^0 c_1^2}{c_1 S_1^0 + c_2 S_2^0} & \dfrac{\beta_{12} p_{12} S_1^0 c_1 c_2}{c_1 S_1^0 + c_2 S_2^0} \\ \dfrac{\beta_{21} p_{21} S_2^0 c_1 c_2}{c_1 S_1^0 + c_2 S_2^0} & -\mu - \gamma_2 + \dfrac{\beta_{22} p_{22} S_2^0 c_2^2}{c_1 S_1^0 + c_2 S_2^0} \end{pmatrix}.$$

We simplify the notation by defining $\delta_i := \mu + \gamma_i$ and $a_{ij} := \frac{\beta_{ij} p_{ij} S_i^0 c_i c_j}{N^0}$. Then

$$J^0 = \begin{pmatrix} -\delta_1 + a_{11} & a_{12} \\ a_{21} & -\delta_2 + a_{22} \end{pmatrix}. \tag{3.6}$$

The larger eigenvalue of J^0,

$$\rho^* := \frac{1}{2}\left(-\left(\delta_1 - a_{11} + \delta_2 - a_{22}\right) + \sqrt{\left(\left(\delta_1 - a_{11}\right) - \left(\delta_2 - a_{22}\right)\right)^2 + 4 a_{12} a_{21}}\right),$$

is real. If $\rho^* < 0$, the zero solution of (3.2) is stable, and if $\rho^* > 0$, it is unstable.

These conditions can be combined to define the reproductive number as

$$R_0 = \frac{1}{(2\mu+\gamma_1+\gamma_2)N^0}\left(\alpha_{11}+\alpha_{22}+\sqrt{(\gamma_1-\gamma_2+\alpha_{22}-\alpha_{11})^2+4\alpha_{12}\alpha_{21}}\right), \tag{3.7}$$

where

$$\alpha_{11} = S_1^0 c_1^2 \beta_{11} w_1(0), \qquad \alpha_{12} = S_1^0 c_1 c_2 \beta_{12} h(0) g(0),$$
$$\alpha_{21} = S_2^0 c_1 c_2 \beta_{21} h(0) g(0), \qquad \alpha_{22} = S_2^0 c_2^2 \beta_{22} w_2(0).$$

Hence, if $R_0 < 1$, the epidemic dies out, and if $R_0 > 1$, the epidemic spreads in the population.

4. Endemic equilibrium and local stability. In this section, we first show that when $R_0 > 0$, there are no boundary equilibria $(I_1 > 0, I_2 = 0)$ and $(I_1 = 0, I_2 > 0)$.

At an equilibrium, the components I_i satisfy the equations

$$c_1\left(S_1^0 - I_1\right)\left(\beta_{11}c_1 w_1(I_1) I_1 + \beta_{12} c_2 h(I_1) g(I_2) I_2\right) = (\mu+\gamma_1) N^0 I_1, \tag{4.1}$$

$$c_2\left(S_2^0 - I_2\right)\left(\beta_{21}c_1 h(I_1) g(I_2) I_1 + \beta_{22} c_2 w_2(I_2) I_2\right) = (\mu+\gamma_2) N^0 I_2. \tag{4.2}$$

If $I_1 = 0$, it follows from (4.1) that

$$c_1 S_1^0 \beta_{12} c_2 h(I_1) g(I_2) I_2 = 0,$$

which implies $I_2 = 0$. A similar argument for $I_2 = 0$ verifies that then I_1 must be zero. Thus there are no boundary equilibria.

To explore the existence of a positive endemic equilibrium, we define

$$F_1(I_1, I_2) := c_1\left(S_1^0 - I_1\right)\left(\beta_{11}c_1 w_1(I_1) I_1 + \beta_{12} c_2 h(I_1) g(I_2) I_2\right) - (\mu+\gamma_1) N^0 I_1$$

for $0 \le I_1 < S_1^0$ and

$$F_2(I_1, I_2) := c\left(S_2^0 - I_2\right)\left(\beta_{21}c_1 h(I_1) g(I_2) I_1 + \beta_{22} c_2 w_2(I_2) I_2\right) - (\mu+\gamma_2) N^0 I_2$$

for $0 \le I_2 < S_2^0$. Then the endemic equilibrium is the positive solution of the system of nonlinear equations $F_1(I_1, I_2) = 0 = F_2(I_1, I_2)$.

We first prove a uniqueness lemma for the equilibrium.

LEMMA 4.1. *The curve $F_1(I_1, I_2) = 0$ or $F_2(I_1, I_2) = 0$ intersects only the ray $I_2 = kI_1$, $I_1 > 0$, at most once for any positive real number $k > 0$.*

Proof. We give only the proof for $F_1(I_1, I_2) = 0$. A similar proof holds for $F_2(I_1, I_2) = 0$.

Any intersection of $F_1(I_1, I_2) = 0$ and $I_2 = kI_1$ must satisfy

$$F_1(I_1, kI_1) = c_1\left(S_1^0 - I_1\right)\left(\beta_{11}c_1 w_1(I_1) I_1 + k\beta_{12} c_2 h(I_1) g(kI_1) I_1\right) - (\mu+\gamma_1) N^0 I_1 = 0.$$

Let

$$G(I_1) := c_1\left(S_1^0 - I_1\right)\left(\beta_{11}c_1 w_1(I_1) + k\beta_{12} c_2 h(I_1) g(kI_1)\right) - (\mu+\gamma_1) N^0.$$

Because

$$\begin{aligned} G'(I_1) = &- c_1\left(\beta_{11}c_1 w_1(I_1) + k\beta_{12} c_2 h(I_1) g(kI_1)\right) \\ &+ c_1\left(S_1^0 - I_1\right)\left(\beta_{11}c_1 w_1'(I_1) + k\beta_{12} c_2\left(h'(I_1) g(kI_1) + k h(I_1) g'(kI_1)\right)\right), \end{aligned}$$

it follows from the assumption $w_i'(I_1) < 0$, $h'(I_1) < 0$, and $g'(I_2) < 0$ that $G'(I_1) < 0$ for $0 \le I_1 < S_1^0$. The conclusion of Lemma 4.1 then immediately follows. □

Using Lemma 4.1, we next prove an existence theorem for the endemic equilibrium.

THEOREM 4.1. *If the reproductive number $R_0 < 1$, there is no positive endemic equilibrium. That is, the infection-free equilibrium is the only equilibrium. If $R_0 > 1$, there exists a unique positive endemic equilibrium such that $0 < S_i^* < S_i^0$ and $0 < I_i^* < S_i^0$.*

Proof. Consider the curve $F_1(I_1, I_2) = 0$. If $I_1 = 0$, then it follows from $F_1(0, I_2) = c_1 c_2 \beta_{12} S_1^0 h(0) g(I_2) I_2 = 0$ that $I_2 = 0$. That is, that the curve $F_1(I_1, I_2) = 0$ crosses the I_2-axis only at the origin. On the other hand,

$$\begin{aligned} 0 &= \lim_{I_1 \to S_1^{0-}} F_1(I_1, I_2) \\ &= \left(\lim_{I_1 \to S_1^{0-}} c_1 \left(S_1^0 - I_1 \right) \right) \beta_{12} c_2 h(S_1^0) \left(\lim_{I_1 \to S_1^{0-}} g(I_2) I_2 \right) - (\mu + \gamma_1) N^0 S_1^0. \end{aligned}$$

This implies that along the curve $F_1(I_1, I_2) = 0$, as I_1 approaches S_1^0, I_2 approaches $+\infty$ from the left. Similarly, we can show that the curve $F_2(I_1, I_2) = 0$ crosses the I_1-axis only at the origin and that I_1 goes to infinity, as $I_2 \to S_2^{0-}$, along the curve $F_2(I_1, I_2) = 0$. That is, the level curves $F_i(I_1, I_2) = 0$ both pass through the origin and go to infinity vertically and horizontally as $I_i \to S_i^{0-}$, respectively.

Assume $R_0 < 1$. Then the infection-free equilibrium is stable, which implies that the two diagonal elements of the Jacobian matrix at the infection-free equilibrium must be negative, and the determinant of the Jacobian must be positive. Then

$$\beta_{ii} w_i(0) \frac{S_i^0 c_i^2}{N^0} - (\mu + \gamma_i) < 0, \qquad i = 1, 2, \tag{4.3}$$

and

$$\left(\beta_{11} w_1(0) \frac{S_1^0 c_1^2}{N^0} - (\mu + \gamma_1) \right) \left(\beta_{22} w_2(0) \frac{S_2^0 c_2^2}{N^0} - (\mu + \gamma_2) \right) > \beta_{12} \beta_{21} h^2(0) g^2(0) \frac{S_1^0 S_2^0 c_1^2 c_2^2}{\left(N^0\right)^2};$$

that is,

$$\frac{c_1^2 S_1^0 \beta_{11} w_1(0) - (\mu + \gamma_1) N^0}{c_1^2 S_1^0 \beta_{12} h(0) g(0)} \frac{c_2^2 S_2^0 \beta_{22} w_2(0) - (\mu + \gamma_2) N^0}{c_2^2 S_2^0 \beta_{21} h(0) g(0)} > 1. \tag{4.4}$$

A simple calculation leads to

$$\begin{aligned} \left. \frac{dI_2}{dI_1} \right|_{F_1(0,0)=0} &= -\frac{c_1^2 S_1^0 \beta_{11} w_1(0) - (\mu + \gamma_1) N^0}{c_1^2 S_1^0 \beta_{12} h(0) g(0)}, \\ \left. \frac{dI_1}{dI_2} \right|_{F_2(0,0)=0} &= -\frac{c_2^2 S_2^0 \beta_{22} w_2(0) - (\mu + \gamma_2) N^0}{c_2^2 S_2^0 \beta_{21} h(0) g(0)}. \end{aligned}$$

Hence, it follows from (4.3) and (4.4) that both slopes of $F_i = 0$ are positive at the origin and that

$$\left. \frac{dI_2}{dI_1} \right|_{F_1(0,0)=0} > \left. \frac{dI_2}{dI_1} \right|_{F_2(0,0)=0} = \frac{1}{\left. \dfrac{dI_1}{dI_2} \right|_{F_2(0,0)=0}},$$

which implies that the slope of the curve $F_1 = 0$ is greater than the slope of $F_2 = 0$ at $(0, 0)$, or the curve $F_1 = 0$ is above the curve $F_2 = 0$ as (I_1, I_2) is near zero.

Suppose that there is an endemic equilibrium in this case; that is, there is an intersection between these two curves in the positive quadrant of the I_1-I_2 plane, say, $(\tilde{I}_1, \tilde{I}_2)$. Then the origin, portions of these two curves, and the point $(\tilde{I}_1, \tilde{I}_2)$ form a closed loop, and a ray from the origin in the positive quadrant intersects either the curve $F_1 = 0$ or F_2 on this closed loop. However, because $F_i = 0$ goes to infinity vertically or horizontally, as the ray goes further, it must cross the curve again, which contradicts the conclusion of Lemma 4.1. Hence, the endemic equilibrium does not exist if $R_0 < 1$.

If $R_0 > 1$, then the diagonal elements of the Jacobian matrix at the infection-free equilibrium can be both negative, one of them negative and the other positive, or both positive. If at least one of them is positive, then the corresponding curve goes to the third quadrant or the fourth quadrant, and the conclusion immediately follows. If both are negative as in (4.3), because $R_0 > 1$, the slope of the curve $F_1 = 0$ is less than the slope of $F_2 = 0$ at $(0, 0)$, or the curve $F_1 = 0$ is below the curve $F_2 = 0$ as (I_1, I_2) near zero. Because, again, the curve $F_1 = 0$ goes to infinity vertically, and the curve $F_2 = 0$ goes to infinity horizontally, there must exist at least one endemic equilibrium. Furthermore, it is unique. Indeed, if there exist two endemic equilibria, these two equilibria and the portions from the curves $F_i = 0$ form a closed loop. Using the same argument as in the case $R_0 < 1$, there exists a ray from the origin in the positive quadrant crossing one of the two curves $F_i = 0$ in this closed loop, and as the ray goes further, it intersects the curve again, which contradicts the conclusion of Lemma 4.1. Therefore, there exists one and only one endemic equilibrium. ∎

The local stability of the positive endemic equilibrium is obtained as follows.

THEOREM 4.2. *If $R_0 > 1$, the unique positive endemic equilibrium is locally stable.*

Proof. The Jacobian matrix of (3.2) at the unique positive endemic equilibrium (I_1^*, I_2^*) is

$$J^* := \begin{pmatrix} -(\mu + \gamma_1 + \lambda_1) + (S_1^0 - I_1^*)\dfrac{\partial \lambda_1}{\partial I_1^*} & (S_1^0 - I_1^*)\dfrac{\partial \lambda_1}{\partial I_2^*} \\ (S_2^0 - I_2^*)\dfrac{\partial \lambda_2}{\partial I_1^*} & -(\mu + \gamma_2 + \lambda_2) + (S_2^0 - I_2^*)\dfrac{\partial \lambda_2}{\partial I_2^*} \end{pmatrix},$$

where λ_i are evaluated at (I_1^*, I_2^*).

Because (I_1^*, I_2^*) is an endemic equilibrium,

$$(\mu + \gamma_i + \lambda_i) I_i^* = \lambda_i S_i^0;$$

that is,

$$\mu + \gamma_i + \lambda_i = \lambda_i \frac{S_i^0}{I_i^*}.$$

Denote the elements of J^* by J_{ij} and write $\xi_{ij} := \beta_{ij} c_j$. Then

$$J_{11} = \frac{c_1}{N^0 I_1^*}\left(-S_1^0 \xi_{12} h g I_2^* + (S_1^0 - I_1^*)\left(\xi_{11} w_1' {I_1^*}^2 + \xi_{12} h' g I_1^* I_2^*\right) - {I_1^*}^2 \xi_{11} w_1\right),$$

$$J_{22} = \frac{c_2}{N^0 I_2^*}\left(-S_2^0 \xi_{21} h g I_1^* + (S_2^0 - I_2^*)\left(\xi_{21} h g' I_1^* I_2^* + \xi_{22} w_2' {I_2^*}^2\right) - {I_2^*}^2 \xi_{22} w_2\right),$$

$$J_{12} = \frac{c_1}{N^0 I_2^*}\left(S_1^0 - I_1^*\right)\left(\xi_{12} hg I_2^* + \xi_{12} hg' {I_2^*}^2\right),$$

$$J_{21} = \frac{c_2}{N^0 I_1^*}\left(S_2^0 - I_2^*\right)\left(\xi_{21} hg I_1^* + \xi_{21} h' g {I_1^*}^2\right),$$

where w_i, h, g, w_i', h', and g' are evaluated at (I_1^*, I_2^*).

Note that every term in J_{11} and J_{22} is negative. Hence, the trace of J^* is negative and then the stability of J^* is determined by the determinant of J^*, $\det J^*$. Now we show $\det J^* > 0$ briefly. Because each term in J_{ii} is negative, we need to find terms in $J_{11}J_{22}$ to cancel the positive terms in $J_{12}J_{21}$.

After factoring out $c_1/(N^0 I_1^*)$ and $c_2/(N^0 I_2^*)$, the only positive terms in $J_{12}J_{21}$ are

$$\left(S_1^0 - I_1^*\right)\left(S_2^0 - I_2^*\right)\xi_{12}\xi_{21}\left(hg\right)^2 I_1^* I_2^* \quad \text{and} \quad \left(S_1^0 - I_1^*\right)\left(S_2^0 - I_2^*\right)\xi_{12}\xi_{21} hgh'g' {I_1^*}^2 {I_2^*}^2.$$

The second term is eliminated by the same term in $J_{11}J_{22}$. We rewrite the first term as

$$S_1^0 S_2^0 \xi_{12}\xi_{21}\left(hg\right)^2 I_1^* I_2^* - \left(S_1^0 I_2^* + I_1^*\left(S_2^0 - I_2^*\right)\right)\xi_{12}\xi_{21}\left(hg\right)^2 I_1^* I_2^*.$$

This term can now be eliminated by a similar term in $J_{11}J_{22}$. Because the remaining terms are all negative, $\det J^* > 0$, and hence the positive endemic equilibrium is locally stable. ∎

5. Global stability. The local stability of the infection-free equilibrium determines the epidemic threshold conditions and determines if a small initial infection spreads. If the endemic equilibrium is stable, then an initial distribution close to the endemic equilibrium will eventually approach the endemic equilibrium. The next step is to determine if the equilibria are globally stable. We first prove that solutions of (3.2) with positive initial values remain positive and bounded.

LEMMA 5.1. *Define the set D as $D := \left\{(I_1, I_2) \in \mathbb{R}^2, \quad 0 \le I_1 \le S_1^0, \quad 0 \le I_2 \le S_2^0\right\}$. Then D is a global attractor for all orbits with positive initial data, and hence the positive quadrant is forward invariant under the flow defined by* (3.2).

Proof. Notice that $\lambda_i > 0$ for all $I_i \ge 0$, $i = 1, 2$, and $I_1 I_2 \ne 0$. Then $dI_i/dt < 0$ if $I_i \ge S_i^0$. Hence D is a global attractor in the positive quadrant. On the boundary $I_i = 0$, $0 < I_j < S_j^0$, $j \ne i$, $dI_i/dt = \lambda_i S_i^0 > 0$, and on the boundary $I_i = S_i^0$, $0 < I_j < S_j^0$, $j \ne i$, $dI_i/dt = -(\mu + \gamma_i + \lambda_i)I_i < 0$. Hence, no orbit in D can ever leave D. ∎

Next we use the Bendixson–Dulac principle [6, 23] to exclude the possibility of the existence of limit cycles or closed-phase polygons in region D.

LEMMA 5.2. *The plan autonomous system* (3.2) *can have no limit cycles or closed-phase polygons in region D.*

Proof. Denote the right-hand sides of (3.2) as P and Q, respectively, and multiply them by $1/I_1 I_2$. Then

$$\frac{\partial}{\partial I_1}\left(\frac{P}{I_1 I_2}\right) = \frac{c_1}{N^0}\left\{-\left(c_1\beta_{11}\frac{w_1(I_1)}{I_2} + c_2\beta_{12}\frac{h(I_1)}{I_1}g(I_2)\right)\right.$$

$$\left. + \left(S_1^0 - I_1\right)\left(c_1\beta_{11}\frac{w_1'(I_1)}{I_2} + c_2\frac{d}{dI_1}\left(\frac{h(I_1)}{I_1}\right)g(I_2)\right)\right\},$$

$$\frac{\partial}{\partial I_2}\left(\frac{Q}{I_1I_2}\right) = \frac{c_2}{N^0}\left\{-\left(c_1\beta_{21}h(I_1)\frac{g(I_2)}{I_2} + c_2\beta_{22}\frac{w_2(I_2)}{I_1}\right)\right.$$
$$\left. + \left(S_2^0 - I_2\right)\left(c_1\beta_{21}h(I_1)\frac{d}{dI_2}\left(\frac{g(I_2)}{I_2}\right) + c_2\beta_{22}\frac{w'(I_2)}{I_1}\right)\right\}.$$

Notice that $w_i'(I_i) < 0$, $i = 1, 2$, $h'(I_1) < 0$, $g'(I_2) < 0$, so that

$$\frac{d}{dI_1}\left(\frac{h(I_1)}{I_1}\right) = \frac{h'(I_1)I_1 - h(I_1)}{I_1^2} < 0,$$
$$\frac{d}{dI_2}\left(\frac{g(I_2)}{I_2}\right) = \frac{g'(I_2)I_2 - g(I_2)}{I_2^2} < 0.$$

The divergence

$$\frac{\partial}{\partial I_1}\left(\frac{P}{I_1I_2}\right) + \frac{\partial}{\partial I_2}\left(\frac{Q}{I_1I_2}\right) < 0$$

along the flow of (3.2) in the interior of region D. Hence, by the Bendixson–Dulac principle there is no limit cycle nor closed-phase polygon in D. ∎

Based on Lemma 5.1 and Lemma 5.2, we have the following theorem.

THEOREM 5.1. *If $R_0 < 1$, the infection-free equilibrium is a global attractor in the positive quadrant. If $R_0 > 1$, the endemic equilibrium is a global attractor in the positive quadrant. That is, for any solution $(I_1(t), I_2(t))$ of (3.2) with $(I_1(0) > 0, I_2(0) > 0)$*

$$R_0 < 1 \implies \lim_{t\to\infty}(I_1(t), I_2(t)) = (0, 0),$$
$$R_0 > 1 \implies \lim_{t\to\infty}(I_1(t), I_2(t)) = (I_1^*, I_2^*),$$

where (I_1^*, I_2^*) is the unique positive endemic equilibrium.

Proof. When $R_0 < 1$, it follows from Theorem 4.1 that the infection-free equilibrium is the only equilibrium in the positive quadrant. By Lemma 5.1, orbits starting in the positive quadrant approach and remain in D for all $t \geq 0$. By Lemma 5.2, there are no limit cycles or closed-phase polygons in D. Then, since the infection-free equilibrium is locally asymptotically stable, it follows from the Poincaré–Bendixson theorem that the ω-limit set Ω must consist solely of this point, which leads to the global stability of the infection-free equilibrium.

Arguing exactly as above, if $R_0 > 1$, because the infection-free equilibrium is unstable and the positive endemic equilibrium is unique, Ω must consist solely of the positive endemic equilibrium, which shows that the positive endemic equilibrium is globally stable. ∎

6. Discussion. The reproductive number and endemic equilibrium can be used to characterize the effects of behavior changes. For example, we can investigate how behavior changes will affect the endemic equilibrium, in particular, how much behavior change would be needed to reduce the reproductive number below the threshold? When the reproductive number is greater than one, it follows from Theorem 5.1 that the positive endemic equilibrium is globally stable. Then no matter how dramatic the changes people make in the selection of their partners as the epidemic spreads, it is not possible to reduce the epidemic below the threshold. That is, a simple switch of

partners between groups or a temporary decrease in the number of partners (w_i, h, and g become smaller as the fraction of infected population is higher), as the epidemic spreads, cannot eradicate the epidemic under the assumption (3.4) (w_i, h, and g are always positive).

Our analysis for the infection-dependent desirability and acceptability can be also applied to the infection-dependent transmission probability, β_{ij}. That is, if we assume that $\beta_{ij} = \beta_{ij}(I_j/N_j) : \mathbb{R} \to \mathbb{R}^+$, are continuously differentiable and decreasing positive functions to characterize behavior changes (such as increased use of condoms as people use information about the infection level to lower their risk of infection from partners in highly infected populations), the general conclusions are the same as for infection-dependent desirability and acceptability functions. Hence, as long as the active individuals keep sexual contacts in any circumstances, once the epidemic threshold has been exceeded, any use of "safe sex" will not eradicate the epidemic.

The only behavior changes that can affect the threshold conditions are those that take place near the infection-free equilibrium. The reproductive number given in (3.7) combines all the demographic and epidemiological parameters and the desirability and acceptability functions evaluated at zero. It is determined by the stability of the infection-free equilibrium where all components $I_i = 0$; this is the situation where none or only a few individuals are infected. From the assumption (3.4), the desirability and acceptability functions are always positive; that is, there are always partnership formations between and within groups. Then, the situation with no individual or a few of individuals infected is the "best situation" in the whole course of the epidemic. Therefore, in order to reduce the infection below the epidemic threshold, people have to make essential behavior changes at or near this "best situation." In other words, people need to reduce their social contacts, c_i, to use "safe sex" to lower the rate of transmission per sexual contact or to keep partnership formations sufficiently low even though there are only a small number of infected individuals in the population.

Our analysis can be used to gain insight into the effects of behavior changes on the dynamics of disease transmission and can be used to provide guidance to sexually active individuals. However, it is unrealistic to expect a dramatic change in the sexually active population, which is partly characterized by $w_i(0)$, $h(0)$, and $g(0)$. In order to provide more significant and more realistic education campaigns, we may advise those people to consider temporarily stopping their partnership formations when the infection becomes severe. This can be modeled by modifying assumption (3.4). Instead of assuming that the desirability and acceptability functions are always positive, we may allow them to be zero when the infection level is above a critical value.

We have also extended this model so that the social contact rates are density dependent and decrease as the fraction of the population infected with the disease increases. The analysis of this and other modified feedback models will help us gain insight into the effect that dynamic behavior changes can have on the course of epidemics.

REFERENCES

[1] M. Becker and J. Joseph, *AIDS and behavioral change to reduce risk: A review*, Amer. J. Public Health, 78 (1988), pp. 394–410.

[2] S. M. Blower and A. R. McLean, *Prophylactic vaccines, risk behavior change, and the probability of eradicating HIV in San Francisco*, Science, 265 (1994), pp. 1451–1454.

[3] S. P. Blythe, C. Castillo-Chavez, J. S. Palmer, and M. Cheng, *Toward a unified theory of sexual mixing and pair formation*, Math. Biosci., 107 (1991), pp. 379–405.

[4] S. Busenberg and C. Castillo-Chavez, *Interaction, pair formation and force of infection terms in sexually transmitted diseases*, in Mathematical and Statistical Approaches to AIDS Epidemiology, Lecture Notes in Biomath. 83, C. Castillo-Chavez, ed., Springer-Verlag, New York, 1989, pp. 289–300.

[5] S. Busenberg and C. Castillo-Chavez, *A general solution of the problem of mixing of subpopulations and its application to risk- and age-structured epidemic models for the spread of AIDS*, IMA J. Math. Appl. Med. Biol., 8 (1991), pp. 1–29.

[6] S. Busenberg and P. van den Driessche, *A method for proving the non-existence of limit cycles*, J. Math. Anal. Appl., 172 (1993), pp. 463–479.

[7] C. Castillo-Chavez and S. P. Blythe, *Mixing framework for social/sexual behavior*, in Mathematical and Statistical Approaches to AIDS Epidemiology, Lecture Notes in Biomath. 83, C. Castillo-Chavez, ed., Springer-Verlag, New York, 1989, pp. 275–288.

[8] C. Castillo-Chavez, K. Cooke, W. Huang, and S. A. Levin, *On the role of long incubation periods in the dynamics of acquired immunodeficiency syndrome, Part* 2: *Multiple group models*, in Mathematical and Statistical Approaches to AIDS Epidemiology, Lecture Notes in Biomath. 83, C. Castillo-Chavez, ed., Springer-Verlag, New York, 1989, pp. 200–217.

[9] C. Castillo-Chavez and H. R. Thieme, *Asymptotically autonomous epidemic models*, in Mathematical Population Dynamics: Proc. Third Internat. Conf. on Mathematical Population Dynamics, O. Arino and M. Kimmel, eds., Marcel Dekker, New York, 1995, pp. 33–50.

[10] Centers for Disease Control, *Patterns of sexual behavior change among homosexual/bisexual men – selected U.S. sites*, 1987–1990, MMWR Morbid. Mortal. Wkly. Rep., 40 (1991), pp. 792–794.

[11] O. Diekmann, J. A. P. Heesterbeek, and J. A. J. Metz, *On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations*, J. Math. Biol., 28 (1990), pp. 365–382.

[12] H. Heesterbeek, R_0, Thesis, Centrum voor Wiskunde en Informatica, Amsterdam, 1992.

[13] H. W. Hethcote, *Qualitative analysis for communicable disease models*, Math. Biosci., 28 (1976), pp. 335–356.

[14] H. W. Hethcote and J. A. Yorke, *Gonorrhea Transmission Dynamics and Control*, Lecture Notes in Biomath. 56, Springer-Verlag, New York, 1984.

[15] *HIV/AIDS Surveillance Data Base*, Center for International Research of the US Bureau of the Census.

[16] J. M. Hyman and Jia Li, *Disease Transmission Models with Biased Partnership Selection*, Report LA-UR-95-2832, Los Alamos National Laboratory, 1995.

[17] J. M. Hyman and E. A. Stanley, *Using mathematical models to understand the AIDS epidemic*, Math. Biosci., 90 (1988), pp. 415–473.

[18] J. M. Hyman and E. A. Stanley, *The effect of social mixing patterns on the spread of AIDS*, in Mathematical Approaches to Problems in Resource Management and Epidemiology, Lecture Notes in Biomath. 81, C. Castillo-Chavez, S. A. Levin, and C. A. Shoemaker, eds., Springer-Verlag, New York, 1989, pp. 190–219.

[19] J. A. Jacquez, C. P. Simon, J. Koopman, L. Sattenspiel, and T. Perry, *Modelling and analyzing HIV transmission: The effect of contact patterns*, Math. Biosci., 92 (1988), pp. 119–199.

[20] J. A. Jacquez, C. P. Simon, and J. Koopman, *Structured mixing: Heterosexual mixing by the definition of activity groups*, in Mathematical and Statistical Approaches to AIDS Epidemiology, Lecture Notes in Biomath. 83, C. Castillo-Chavez, ed., Springer-Verlag, New York, 1989, pp. 301–315.

[21] J. A. Jacquez, C. P. Simon, and J. Koopman, *The reproductive number in deterministic models of contagious diseases*, Comm. Theor. Biol., 2 (1991), pp. 159–209.

[22] A. M. Johnson and O. N. Gill, *Evidence for recent changes in sexual behavior in homosexual men in England and Wales*, Philos. Trans. Roy. Soc. London Biol., 325 (1989), pp. 153–161.

[23] D. W. Jordan and P. Smith, *Nonlinear Ordinary Differential Equations*, Oxford University Press, New York, 1987.

[24] J. S. Koopman, C. P. Simon, J. A. Jacquez, and T. S. Park, *Selective contact within structured mixing with an application to HIV transmission risk from oral and anal sex*, in Mathematical and Statistical Approaches to AIDS Epidemiology, Lecture Notes in Biomath. 83, C. Castillo-Chavez, ed., Springer-Verlag, New York, 1989, pp. 317–348.

[25] J. S. Koopman, I. M. Longini Jr., J. A. Jacquez, C. P. Simon, D. G. Ostrow, W. R. Martin, and D. M. Woodcock, *Assessing risk factors for transmission of infection*, Amer. J. Epidemiol., 133 (1991), pp. 1199–1209.

[26] Jia Li, *Effects of risk behavior change on the spread of AIDS epidemic*, Math. Comput. Modelling, 16 (1992), pp. 103–111.

[27] J. L. Martin, *The impact of AIDS on gay male sexual behavior patterns in New York City*, Amer. J. Public Health, 77 (1987), pp. 578–581.
[28] L. McKusick, W. Horstman, and T. Coastes, *AIDS and sexual behavior reported by gay men in San Francisco*, Amer. J. Public Health, 75 (1985), pp. 493–496.
[29] A. R. McLean and S. M. Blower, *Imperfect vaccines and herd immunity to HIV*, Proc. Roy. Soc. London Ser. B, 253 (1993), pp. 9–13.
[30] M. Morris and L. Dean, *Effects of sexual behavior change on long-term human immunodeficiency virus prevalence among homosexual men*, Amer. J. Epidemiol., 140 (1994), pp. 217–232.
[31] L. Sattenspiel, *Population structure and the spread of disease*, Human Biol., 59 (1987), pp. 411–438.
[32] L. Sattenspiel, *Epidemics in nonrandomly mixing populations: A simulation*, Am. J. Physical Anthropol., 73 (1987), pp. 251–265.
[33] L. Sattenspiel and C. Castillo-Chavez, *Environmental context, social interaction, and the spread of HIV*, Am. J. Human Biol., 2 (1990), pp. 397–417.
[34] G. Scalia-Tomba, *The effects of structured behavior change on the spread of HIV in a one-sex population*, Math. Biosci., 107 (1991), pp. 547–555.
[35] C. P. Simon and J. A. Jacquez, *Reproductive Numbers and the Stability of Equilibria of SI Models for Heterogeneous Populations*, IMA Preprint Series 669, University of Minnesota, 1990.
[36] H. R. Thieme, *Asymptotically autonomous differential equations in the plane*, Rocky Mountain J. Math., 24 (1994), pp. 351–380.
[37] E. J. C. van Ameijden, J. A. R. van den Hoek, C. Hartgers, and R. A. Coutinho, *Risk factors for the transition from noninjection to injection drug use and accompanying AIDS risk behavior in a cohort of drug users*, Amer. J. Epidemiol., 139 (1994), pp. 1153–1163.
[38] J. X. Velasco-Hernandez and Y. H. Hsieh, *Modelling the effects of treatment and behavioral change in HIV transmission dynamics*, J. Math. Biol., 32 (1994), pp. 233–249.
[39] P. Waltman, *Deterministic Threshold Models in the Theory Epidemics*, Springer-Verlag, New York, 1974.
[40] W. Winkelstein, J. Wiley, N. Padian, et al., *The San Francisco men's health study: Continued decline in HIV seroconversion rates among homosexual/bisexual men*, Amer. J. Public Health, 78 (1988), pp. 1472–1474.